REMARKS / ARGUMENTS

Claim 1 is amended to delete prodrugs of the FLT-3 inhibitor. Claim 3 is cancelled. Claims 5 and 6 are amended so that they are no longer dependent on cancelled claim 3.

Claim 1 is further amended to limit the disease state to those comprising cells that express constitutively active mutant FLT-3. The amendment is supported by the specification, for example at page 49, penultimate paragraph and from page 49, penultimate paragraph, to page 55.

New claim 24 is supported by the specification, particularly by original claims 2, 6 and 14 and the disclosure from page 49, penultimate paragraph, to page 55.

Applicants submit that the amended claims here presented overcome the rejection under 35 USC 112, first paragraph, by eliminating stausporine derivatives of undefined structure and by eliminating the term "prodrug thereof" from the claims. Accordingly, Applicants request withdrawal of the rejection under 35 USC 112, first paragraph.

Claims 1-14 were rejected under 35 USC 103(a) as being unpatentable over Remiszewski et al in view of Verner et al and Griffin et al. Applicants request reconsideration and withdrawal of the rejection for the reasons that follow.

Griffin at al discloses that the present staurosporine derivatives have activity against mutated FLT-3 and discloses AML as a disease characterized by mutated FLT-3. Remiszweski et al and Verner et al contain general disclosures relating to the use of HDAI compounds for the treatment of proliferative diseases, including leukemias, and generally that the HDAI compounds could be used in combination with other therapeutic agents. However, the publications do not provide a basis for one of skill to expect the HDAI to have utility for the treatment of a disease characterized by mutated FLT-3. Therefore, the references do not provide a basis to treat the diseases included within the scope of the present claims with the present combination of therapeutic agents.

In addition, Applicants direct the Examiner's attention to Bali et al, Clinical Cancer Research, Vol. 10, pp. 4991-4997 (2004), which is of record. This publication, which is not prior art and includes the present inventors as authors, discloses a theoretical basis for testing the present combination in diseases characterized by mutated FLT-3. Applicants assert that the combined disclosure of the references would not have led one of skill to the hypothesis and that